WEST Search History

Hide Items | Restore | Clear | Cancel

DATE: Wednesday, March 10, 2004

Hide?	Set Name	Query	Hit Count
	DB=PGPB,USPT,EPAB,JPAB,DWPI; PLUR=YES; OP=OR		
☐	L9	L8 and l7	95
☐	L8	l2 and reactivat$ same (HSV$3 or herpes adj simplex)	304
☐	L7	L6 not l1	132
☐	L6	L5 and (inhibit$ or prevent$ or treat$ or reduc$) same (reactivat$5)	133
☐	L5	(HSV$3 or herpes adj simplex) and reactivat$5 same (model or mouse)	199
☐	L4	L3 and (scratch or abasion or dermabrasion) same (mouse rabbit animal monkey model)	2
☐	L3	L2 not l1	900
☐	L2	(HSV$3 or herpes adj simplex) and reactivat$5 and (model or mouse)	904
☐	L1	(mouse rabbit animal monkey model) same (scratch $4abras$4) same (herpes or HSV$3)	35

END OF SEARCH HISTORY

STN Search History

FILE 'HOME' ENTERED AT 15:10:46 ON 10 MAR 2004

L1 QUE (HSV## OR HERPES (A) SIMPLEX) (P) (REACTIVAT##### OR RECRUD########)

L4 456 L3 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IRRADIAT#####)

L6 518 L1 AND (HSV## OR HERPES) (S) (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IRRADIAT#####)

L9 523 L2 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IRRADIAT#####) (S) (REACTIVAT##### OR RECRUD##########)

(FILE 'HOME' ENTERED AT 15:10:46 ON 10 MAR 2004)

INDEX 'ADISCTI, ADISINSIGHT, ADISNEWS, AGRICOLA, ANABSTR, AQUASCI, BIOBUSINESS, BIOCOMMERCE, BIOSIS, BIOTECHABS, BIOTECHDS, BIOTECHNO, CABA, CANCERLIT, CAPLUS, CEABA-VTB, CEN, CIN, CONFSCI, CROPB, CROPU, DISSABS, DDFB, DDFU, DGENE, DRUGB, DRUGMONOG2, ...' ENTERED AT 15:11:11 ON 10 MAR 2004

L1 QUE (HSV## OR HERPES (A) SIMPLEX) (P) (REACTIVAT##### OR RECRUD

FILE 'MEDLINE, CAPLUS, BIOSIS, EMBASE, LIFESCI, SCISEARCH' ENTERED AT 15:13:32 ON 10 MAR 2004

L2 6038 S L1
L3 3800 S L2 AND (MOUSE OR MURINE OR ANIMAL OR MODEL OR RABBIT)
L4 456 S L3 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IR
L5 204 DUP REM L4 (252 DUPLICATES REMOVED)
L6 518 S L1 AND (HSV## OR HERPES) (S) (UV OR ULTRA-VIOLET OR ULTRVIOLE
L7 149 S L6 AND L5
L8 131 S L7 NOT PY>1999
L9 523 S L2 AND (UV OR ULTRA-VIOLET OR ULTRVIOLET OR RADIAT#### OR IR
L10 115 S L9 AND L8
L11 115 DUP REM L10 (0 DUPLICATES REMOVED)
L12 0 S L11 AND (SCRATCH OR ABRAS######)
L13 6 S L2 AND (SCRATCH OR ABRAS######)
L14 105 S L10 AND (HSV## OR HERPES) (S) (REACTIVAT#####)
L15 80 S L14 NOT PY>1990
L16 35 S L10 NOT L15
L17 80 S L15 NOT L16
L18 62 S L15 AND (HSV## OR HERPES)/AB
L19 43 S L15 AND (HSV## OR HERPES)/TI

L16 ANSWER 3 OF 35 MEDLINE on STN
AN 1999138953 MEDLINE
DN PubMed ID: 9971753
TI **Reactivation** of **herpes simplex** virus type 1 in the **mouse** trigeminal ganglion: an in vivo study of virus antigen and cytokines.
AU Shimeld C; Easty D L; Hill T J
CS Departments of Ophthalmology, University of Bristol, Bristol BS8 1TD, United Kingdom.. C.Shimmeld@bris.ac.uk
SO Journal of virology, (1999 Mar) 73 (3) 1767-73.
Journal code: 0113724. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199903
ED Entered STN: 19990316
Last Updated on STN: 19990316
Entered Medline: 19990304
AB **Reactivation** of **herpes simplex** virus type 1 (**HSV**-1) in the trigeminal ganglion (TG) was induced by **UV irradiation** of the corneas of latently infected **mice**. Immunocytochemistry was used to monitor the dynamics of cytokine (interleukin-2 [IL-2], IL-4, IL-6, IL-10, gamma interferon [IFN-gamma], and tumor necrosis factor alpha [TNF-alpha]) and viral antigen production in the TG and the adjacent central nervous system on days 1 to 4, 6, 7, and 10 after **irradiation**. **UV irradiation** induced increased expression of IL-6 and TNF-alpha from satellite cells in uninfected TG. In latently infected TG, prior to **reactivation**, all satellite cells were TNF-alpha+ and most were also IL-6(+). **Reactivation**, evidenced by **HSV**-1 antigens and/or infiltrating immune cells, occurred in 28 of 45 (62%) TG samples. Viral antigens were present in the TG in neurons, often disintegrating on days 2 to 6 after **irradiation**. Infected neurons were usually surrounded by satellite cells and the foci of immune cells producing TNF-alpha and/or IL-6. IL-4(+) cells were detected as early as day 3 and were more numerous by day 10 (a very few IL-2(+) and/or IFN-gamma+ cells were seen at this time). No IL-10 was detected at any time. Our observations indicate that **UV irradiation** of the cornea may modulate cytokine production by satellite cells. We confirm that neurons are the site of **reactivation** and that they probably do not survive this event. The predominance of TNF-alpha and IL-6 following **reactivation** parallels primary infection in the TG and suggests a role in viral clearance. The presence of Th2-type cytokines (IL-4 and IL-6) indicates a role for antibody. Thus, several clearance mechanisms may be at work.

L16 ANSWER 6 OF 35 MEDLINE on STN
AN 97042294 MEDLINE
DN PubMed ID: 8887494
TI **Reactivation** of **herpes simplex** virus type 1 in the **mouse** trigeminal ganglion: an in vivo study of virus antigen and immune cell infiltration.
CM Erratum in: J Gen Virol 1996 Dec;77(Pt 12):3165
AU Shimeld C; Whiteland J L; Williams N A; Easty D L; Hill T J
CS Department of Ophthalmology, School of Medical Sciences, Bristol, UK.. C.Shimeld@bris.ac.uk
SO Journal of general virology, (1996 Oct) 77 (Pt 10) 2583-90.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199612
ED Entered STN: 19970128
Last Updated on STN: 19980206
Entered Medline: 19961213
AB The corneas of latently infected **mice** were **UV irradiated** to induce **reactivation** of **herpes simplex** virus type 1 (**HSV**-1) in the trigeminal ganglion (TG). On days 1 to 4 after **irradiation**, TG were removed, serially sectioned and double stained to identify immune cells and virus antigens. Virus antigen was detected in small numbers (most commonly one) of neurons per ganglion as early as day 1, confirming the rapidity of **reactivation** and the neuron as the likely site of this event. The immune response was also rapid and effective since virus antigen was identified in immune cells at day 1 and by day 4 all samples were negative. The predominant infiltrating cells on days 1 and 2, when virus antigen was present and being cleared, were T cells, both CD4+ and CD8+. Later, large numbers of B cells appeared, suggesting that local antibody production may also be involved in controlling the **reactivated** infection. The observations suggest that a significant proportion of **reactivation** events do not result in disease of the eye or shedding of virus in the tear film. However, they also suggest that as little as one **reactivating** neuron in the ganglion may be sufficient to lead to such disease and/or shedding.

L16 ANSWER 8 OF 35 MEDLINE on STN
AN 96336924 MEDLINE
DN PubMed ID: 8760562
TI **UV radiation** and **mouse models** of **herpes** simplex virus infection.
AU Norval M; el-Ghorr A A
CS Department of Medical Microbiology, University of Edinburgh Medical School, Scotland.. M.Norval@ed.ac.uk
SO Photochemistry and photobiology, (1996 Aug) 64 (2) 242-5. Ref: 26
Journal code: 0376425. ISSN: 0031-8655.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199610
ED Entered STN: 19961015
Last Updated on STN: 19961015
Entered Medline: 19961001
AB Orolabial human infections with **herpes simplex** virus type 1 (**HSV**-1) are very common; following the primary epidermal infection, the virus is retained in a latent form in the trigeminal ganglia from where it can **reactivate** and cause a **recrudescent** lesion. Recrudescences are triggered by various stimuli including exposure to sunlight. In this review three categories of **mouse models** are used to examine the effects of **UV irradiation** on **HSV** infections: these are **UV** exposure prior to primary infection, **UV** exposure as a triggering event for **recrudescence** and **UV** exposure prior to challenge with virus in **mice** already immunized to **HSV**. In each of these **models** immunosuppression occurs, which is manifest, in some instances, in increased morbidity or an increased rate of **recrudescence**. Where known, the immunological

mechanisms involved in the **models** are summarized and their relevance to human infections considered.

L16 ANSWER 10 OF 35 MEDLINE on STN
AN 93389452 MEDLINE
DN PubMed ID: 8397285
TI Effect of indomethacin on ultraviolet **radiation**-induced recurrent **herpes** simplex virus disease in guinea-pigs.
AU Bratcher D F; Harrison C J; Bourne N; Stanberry L R; Bernstein D I
CS Division of Infectious Diseases, Children's Hospital Research Foundation, Cincinnati, Ohio 45229.
NC AI 22667 (NIAID)
AI 23482 (NIAID)
AI 29687 (NIAID)
SO Journal of general virology, (1993 Sep) 74 (Pt 9) 1951-4.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199310
ED Entered STN: 19931105
Last Updated on STN: 19970203
Entered Medline: 19931018
AB Exposure to u.v. **radiation** increases the local level of prostaglandins which may play a role in u.v. **radiation**-induced **herpes simplex** virus (**HSV**) recurrences. We used the guinea-pig **model** of u.v. **radiation**-induced recurrent genital **HSV**-2 disease for examining the effects of indomethacin, a prostaglandin inhibitor, on u.v.-induced recurrences. In the first experiment, performed 100 days after **HSV**-2 inoculation, treatment with indomethacin for 5 days begun 24 h before u.v.-**irradiation** decreased the proportion of **animals** developing **HSV** disease recurrences from 11/13 (84.6%) to 2/13 (15.4%) ($P < 0.001$). In the second experiment, performed 135 days after **HSV**-2 inoculation, treatment with indomethacin for 5 days begun 24 h before u.v.-**irradiation** decreased the number of **animals** developing recurrences from 12/21 (57.1%) to 5/21 (23.8%) ($P < 0.05$). Five days of indomethacin treatment begun 4 h after u.v.-**irradiation**, however, did not reduce the percentage of **animals** developing disease recurrences but did decrease the mean number of days with recurrent lesions in **animals** that developed recurrences. Our data suggest that indomethacin may modify u.v. **radiation**-induced recurrent lesions by decreasing viral **reactivation** when given before u.v. **radiation** exposure or by reducing prostaglandin-induced immunosuppression when given before or after exposure. Future studies are needed for evaluating indomethacin prophylaxis for recurrent **HSV** disease when prolonged u.v. **radiation** exposure is anticipated.

L16 ANSWER 12 OF 35 MEDLINE on STN
AN 92364375 MEDLINE
DN PubMed ID: 1323616
TI **UV** light-induced **reactivation** of **herpes simplex** virus type 2 and prevention by acyclovir.
AU Rooney J F; Straus S E; Mannix M L; Wohlenberg C R; Banks S; Jagannath S; Brauer J E; Notkins A L
CS Laboratory of Oral Medicine, National Institute of Dental Research, National Institutes of Health, Bethesda, MD 20892.
SO Journal of infectious diseases, (1992 Sep) 166 (3) 500-6.
Journal code: 0413675. ISSN: 0022-1899.

CY United States
DT (CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 199209
ED Entered STN: 19920925
Last Updated on STN: 19960129
Entered Medline: 19920917
AB **UV** B light is a potent stimulus for inducing **reactivation** of latent **herpes simplex** virus (**HSV**) infections. Patients were enrolled in a double-blind placebo-controlled crossover trial to determine whether acyclovir can prevent **UV** light-induced **HSV**-2 recurrences. Twenty-four patients with a history of recurrent infection of perigenital sites (e.g., buttock, thigh) were exposed one to four times with 4 minimum erythema doses of **UV** light. Patients were given acyclovir 200 mg orally five times daily or matched placebo beginning 1 day before each exposure and continuing for 5 days after exposure. There were 13 **UV**-induced recurrences among 36 placebo treatments and 3 after 38 acyclovir treatments (P = .004). The mean time to recurrence (+/- SE) was 4.8 +/- 0.3 days. **HSV**-2 lesions developed primarily at the site of **UV** exposure. The cutaneous distribution and timing of **UV**-induced recurrences was consistent with a neural localization (dorsal root ganglia) of latent viral infection. This **UV** light **model** permits direct examination of events leading to **HSV** -2 recurrences in humans and can be used to evaluate approaches to prevention.

L16 ANSWER 14 OF 35 MEDLINE on STN
AN 91373115 MEDLINE
DN PubMed ID: 1654309
TI Characterization of a **murine model** of recurrent **herpes** simplex viral keratitis induced by ultraviolet B **radiation**.
AU Laycock K A; Lee S F; Brady R H; Pepose J S
CS Department of Ophthalmology and Visual Science, Washington University School of Medicine, St. Louis, Missouri 63110.
SO Investigative ophthalmology & visual science, (1991 Sep) 32 (10) 2741-6. Journal code: 7703701. ISSN: 0146-0404.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199110
ED Entered STN: 19911108
Last Updated on STN: 19911108
Entered Medline: 19911024
AB The authors characterized a **murine model** of **herpes simplex** virus (**HSV**) **reactivation** in which recurrent herpetic keratitis was obtained in up to 80% of **animals**. Five weeks after ganglionic latency was established in National Institutes of Health inbred **mice** after corneal inoculation, **HSV** type 1 (**HSV**-1) was **reactivated** by **irradiating** the previously inoculated eye with ultraviolet (**UV**) light. Comparison of different **UV** wavelengths showed UVB to be optimal for **reactivation**, with peak viral recurrence being induced by a total exposure of approximately 250 mJ/cm2. **Reactivated** infectious virus generally began to appear

in trigeminal ganglia 2 days postirradiation and was subsequently detectable in the cornea by both corneal swabbing and immunostaining for viral antigens. Two consecutive outbreaks of viral recurrence at the ocular surface were induced in selected **animals** by serial exposure to UVB. Advantages of this **model** over other **models** of recurrent keratitis are discussed.

L16 ANSWER 15 OF 35 MEDLINE on STN
AN 88034956 MEDLINE
DN PubMed ID: 2822847
TI A **murine model** of **herpes simplex** virus **recrudescence**.
AU Norval M; Howie S E; Ross J A; Maingay J P
CS Department of Bacteriology, University of Edinburgh Medical School, U.K.
SO Journal of general virology, (1987 Oct) 68 (Pt 10) 2693-8.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198711
ED Entered STN: 19900305
Last Updated on STN: 19900305
Entered Medline: 19871125
AB A **murine model** is described in which **recrudescence** of **herpes simplex** virus (**HSV**) type 1 was achieved. C3H **mice** were shaved and **irradiated** with u.v. B light 3 days before being infected epidermally with a clinical isolate of **HSV**. Seven weeks or longer following the primary infection, the survivors were again shaved, **irradiated** with u.v. and mildly tape-stripped. **Recrudescent** lesions occurred in up to 80% of **mice** at the site of the original lesion in most cases, but also occasionally at other sites. Skin painting with u.v.-**irradiated** urocanic acid (a substance suggested to be a photomediator of the immunosuppressive effects of u.v.) in place of u.v.-**irradiation** induced some **recrudescence** but was not as efficient as u.v.-**irradiation** . Antibody titres to **HSV** had no value in predicting whether **recrudescence** would occur but lymphoproliferative responses in draining lymph nodes may provide some indication of viral activity at the epidermal site. A hypothesis is developed that u.v.-**irradiation** before primary infection with **HSV** induces a suppressive immune response to the virus which affects the virus-host interaction and accounts for a high incidence of **recrudescent** lesions on subsequent stimulus.

L16 ANSWER 16 OF 35 MEDLINE on STN
AN 80187958 MEDLINE
DN PubMed ID: 6246417
TI Repair and mutagenesis of **herpes** simplex virus in **UV-irradiated** monkey cells.
AU Lytle C D; Goddard J G; Lin C H
SO Mutation research, (1980 Apr) 70 (2) 139-49.
Journal code: 0400763. ISSN: 0027-5107.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198007
ED Entered STN: 19900315
Last Updated on STN: 19900315

Entered Medline: 19800726

AB Mutagenic repair in mammalian cells was investigated by determining the mutagenesis of **UV-irradiated** or unirradiated **herpes simplex** virus in **UV-irradiated** CV-1 monkey kidney cells. These results were compared with the results for **UV**-enhanced virus **reactivation** (UVER) in the same experimental situation. High and low multiplicities of infection were used to determine the effects of multiplicity **reactivation** (MR). UVER and MR were readily demonstrable and were approximately equal in amount in an infectious center assay. For this study, a forward-mutation assay was developed to detect virus mutants resistant to iododeoxycytidine (ICdR), probably an indication of the mutant virus being defective at its thymidine kinase locus. ICpR-resistant mutants did not have a growth advantage over wild-type virus in irraidated or unirradiated cells. Thus, higher fractions of mutant virus indicated greater mutagenesis during virus repair and/or replication. The data showed that: (1) unirradiated virus was mutated in unirradiated cells, providing a background level of mutagenesis; (2) unirradiated virus was mutated about 40% more in **irradiated** cells, indicating that virus replication (DNA synthesis?) became mutagenic as a result of cell **irradiation**; (3) **irradiated** virus was mutated much more (about 6-fold) than unirradiated virus, even in unirradiated cells; (4) cell **irradiation** did not change the mutagenesis of **irradiated** virus except at high multiplicity of infection. High multiplicity of infection did not lead to higher mutagenesis in unirradiated cells. Thus the data did not demonstrate UVER or MR alone to be either error-free or error-prone. When the two processes were present simultaneously, they were mutagenic.

L16 ANSWER 17 OF 35 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1999:585614 CAPLUS
DN 131:346171
TI Treatment of **HSV**-1 infection with immunoglobulin or acyclovir: comparison of their effects on viral spread, latency, and **reactivation**
AU LeBlanc, Rona A.; Pesnicak, Lesley; Godleski, Matthew; Straus, Stephen E.
CS Medical Virology Section, Laboratory of Clinical Investigation, National Institutes of Health, Bethesda, MD, 20892-1888, USA
SO Virology (1999), 262(1), 230-236
CODEN: VIRLAX; ISSN: 0042-6822
PB Academic Press
DT Journal
LA English
AB We compared Ig (IgG) and acyclovir (ACV) therapies on the establishment, maintenance, and **reactivation** from latency of **HSV**-1(McKrae) in a **mouse** ocular infection **model**. **Mice** were given one i.p. (IP) dose of human IgG 24 h after infection (Day 1 p.i.) or ACV in the drinking water from Days 1 to 7 p.i. Both treatments allowed similar percentages of **mice** to survive the infection and decreased ocular virus shedding as compared with untreated controls. At most time points, there were no differences between IgG- and ACV-treated **animals** with respect to tissue virus titers or in the rates of virus **reactivation** during explant cocultivation. However, after **UV** exposure, **HSV** **reactivated** in 30% of ACV-treated **mice** compared with 90% of IgG-treated **mice** ($P = 0.02$). Also by quant. PCR, we found more latent **HSV**-1 DNA copies in IgG-treated **mice** compared with those given ACV ($P = 0.02$). IgG treatment protects **mice** from **HSV**-1 infection essentially as well as ACV does. Nonetheless, it permits higher levels of latent infection and subsequent in vivo **reactivation**. These studies have

implications for the mechanism by which IgG functions to attenuate **HSV** infections and for its potential value as a therapeutic agent in humans. (c) 1999 Academic Press.

RE.CNT 30 THERE ARE 30 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L16 ANSWER 29 OF 35 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1991:244618 BIOSIS
DN PREV199140118783; BR40:118783
TI NERVE GROWTH FACTOR DOES NOT SUPPRESS **UV**-B INDUCED **REACTIVATION** OF LATENT **HERPES SIMPLEX** VIRUS IN-VIVO.
AU PEPOSE J S [Reprint author]; BRADY R H; LAYCOCK K A; OSBORNE P A; JOHNSON E M JR
CS DEP OPHTHALMOL, WASH UNIV SCH MED, ST LOUIS, MO, USA
SO Investigative Ophthalmology and Visual Science, (1991) Vol. 32, No. 4, pp. 853.
Meeting Info.: ANNUAL SPRING MEETING OF THE ASSOCIATION FOR RESEARCH IN VISION AND OPHTHALMOLOGY, SARASOTA, FLORIDA, USA, APRIL 28-MAY 3, 1991.
INVEST OPHTHALMOL VISUAL SCI.
CODEN: IOVSDA. ISSN: 0146-0404.
DT Conference; (Meeting)
FS BR
LA ENGLISH
ED Entered STN: 21 May 1991
Last Updated on STN: 21 May 1991

L19 ANSWER 2 OF 43 MEDLINE on STN
AN 88307442 MEDLINE
DN PubMed ID: 2841857
TI Cutaneous **herpes** simplex virus lesions induced by ultraviolet **radiation**. A review of **model** systems and prophylactic therapy with oral acyclovir.
AU Spruance S L
CS Department of Medicine, University of Utah School, Salt Lake City, Utah 84132.
SO American journal of medicine, (1988 Aug 29) 85 (2A) 43-5. Ref: 25
Journal code: 0267200. ISSN: 0002-9343.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198809
ED Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880922
AB **Animal** and human **models** of ultraviolet **radiation**-induced **herpes simplex** virus disease provide opportunities to study the mechanism of virus latency and **reactivation**. These **models** can also be used to study the efficacy of antiviral agents. Prophylactic oral acyclovir altered the development of ultraviolet **radiation**-induced **herpes** labialis under both natural and experimental conditions.

L19 ANSWER 7 OF 43 MEDLINE on STN
AN 83141569 MEDLINE
DN PubMed ID: 6298618
TI Enhanced survival of ultraviolet-**irradiated herpes** simplex virus in cells exposed to antiviral agents.
AU Schnipper L E; Lewin A A; Crumpacker C S
NC AI-52530 (NIAID)
NIA-1-P01-AG0059 (NIA)
SO Mutation research, (1983 Feb) 116 (2) 65-72.
Journal code: 0400763. ISSN: 0027-5107.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198304
ED Entered STN: 19900318
Last Updated on STN: 19970203
Entered Medline: 19830415
AB Enhanced survival of **UV-irradiated HSV**-1 is demonstrated in monkey cells exposed to inhibitors of viral DNA synthesis. Phosphonoacetic acid (PAA), adenine arabinoside (ara-A), and cytosine arabinoside (ara-C) pretreatment of infected cells is associated with concentration-dependent **reactivation** of **UV-HSV** -1. At concentrations that result in enhanced virus survival, inhibition of cell DNA synthesis is observed by either ara-A or ara-C, but not by PAA. Pretreatment of uninfected cells with acycloguanosine (ACG) is not associated with **reactivation** of **irradiated HSV** -1, and this is probably due to insufficient generation of ACG-triphosphate, the active inhibitor of viral and cell DNA synthesis.

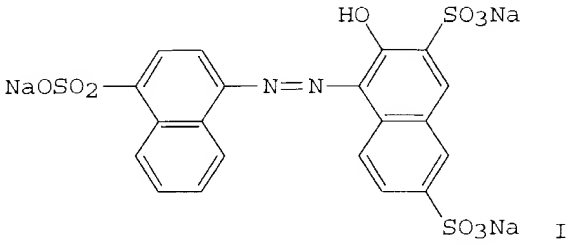
L19 ANSWER 11 OF 43 MEDLINE on STN

AN 80051198 MEDLINE
DN PubMed ID: 227991
TI Acute and latent infection of sensory ganglia with **herpes simplex** virus: immune control and virus **reactivation**.
AU Openshaw H; Asher L V; Wohlenberg C; Sekizawa T; Notkins A L
SO Journal of general virology, (1979 Jul) 44 (1) 205-15.
Journal code: 0077340. ISSN: 0022-1317.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 198001
ED Entered STN: 19900315
Last Updated on STN: 19900315
Entered Medline: 19800124
AB The role of antiviral antibody in controlling the acute and latent phases of **herpes simplex** virus (**HSV**) infection in sensory ganglia of **mice** was studied in vitro and in vivo. Organ cultures of ganglia inoculated in vitro with **HSV** produced infectious virus for at least 3 weeks. In the presence of antiviral antibody, the titre of virus was markedly reduced, but the infection was not eliminated. Similarly, passive administration of antibody to **HSV**-infected immunodeficient (nude) **mice** reduced the virus titre but did not eliminate the acute phase of the ganglionic infection. Suppression of the cell-mediated immune response in latently infected immunocompetent **mice** by treatment with cyclophosphamide and/or X-**irradiation** resulted in **reactivation** of **HSV** in up to 70% of the **animals**. **Reactivation** was demonstrated by recovering infectious virus in cell-free homogenates of ganglia and eye globes and by finding virus antigens in ganglia by immunofluorescent staining. **Reactivation** occurred both in vitro and in vivo in the presence of high concentrations of neutralizing antibody. It is concluded that antibody alone is not sufficient to eliminate the acute phase of the ganglionic infection and that cytotoxic agents known to suppress the host's cellular immune response can **reactivate** virus in the presence of neutralizing antibody.

L19 ANSWER 13 OF 43 MEDLINE on STN
AN 74083359 MEDLINE
DN PubMed ID: 4359192
TI Multiplicity **reactivation** in **UV-irradiated** **herpes simplex** type 1 virus.
AU Roubal J; Vonka V
SO Intervirology, (1973) 1 (2) 73-9.
Journal code: 0364265. ISSN: 0300-5526.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 197403
ED Entered STN: 19900310
Last Updated on STN: 19900310
Entered Medline: 19740320

L19 ANSWER 17 OF 43 CAPLUS COPYRIGHT 2004 ACS on STN
AN 1986:30038 CAPLUS
DN 104:30038
TI Influence of environmental substances on the **reactivation** of **HSV**-2 from latent infection systems in vitro
AU Fukuma, Mariko; Seto, Yoshiko; Toyoshima, Shigeshi
CS Sch. Med., Keio Univ., Tokyo, 160, Japan

SO Chemotherapy (Tokyo) (1985), 33(9), 733-42
CODEN: NKRZAZ; ISSN: 0369-4682
DT Journal
LA Japanese
GI



I

AB Amaranth (I) [915-67-3], erythrosine [16423-68-0], safrole [94-59-7], phenacetin [62-44-2], 2-acetylaminofluorene [53-96-3], SbCl3, and HgCl2 inhibited the **reactivation** of **UV-irradiated herpes simplex** virus type 2 (**HSV**-2) from some latent infection system. Nicotine [54-11-5] enhanced the **reactivation** of **UV-irradiated HSV**-2 at a nonpermissive temperature (NPT) to a permissive one, but it did not enhance the **reactivation** in an Ara C-treated cell system. None of the compds. **reactivated HSV**-2 from the latent state at an NPT without any **reactivation**. Clear results could not be obtained in the trigeminal ganglion system because of a large variation of virus yield **reactivated**. Thus, some environmental substances may influence the onset and cure of recurrent **HSV** infection.

L19 ANSWER 31 OF 43 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1981:236262 BIOSIS
DN PREV198172021246; BA72:21246
TI LATENCY IN-VITRO USING **IRRADIATED HERPES** SIMPLEX VIRUS.
AU NISHIYAMA Y [Reprint author]; RAPP F
CS DEP MICROBIOL, PA STATE UNIV COLL MED, HERSHEY, PA 17033 USA, USA
SO Journal of General Virology, (1981) Vol. 52, No. 1, pp. 113-120.
CODEN: JGVIAY. ISSN: 0022-1317.
DT Article
FS BA
LA ENGLISH
AB Human embryonic fibroblasts infected with **UV-irradiated herpes simplex** virus type 2 (**HSV**-2, strain 186) and maintained at 40.5° C did not yield detectable virus. Virus synthesis was induced by temperature shift-down to 36.5° C. The induced virus grew very poorly and was inactivated very rapidly at 40.5° C. Non-**irradiated** virus failed to establish latency at 40.5° C in infected cells. Enhanced **reactivation** of **HSV**-2 was observed when latently infected cultures were superinfected with human cytomegalovirus (HCMV) or **irradiated** with a small dose of **UV** light at the time of temperature shift-down. HCMV did not enhance synthesis of **HSV**-2 during a normal growth cycle but did enhance synthesis of **UV-irradiated HSV**-2. In this in vitro latency system, some **HSV** genomes damaged by **UV irradiation** were

maintained in a non-replicating state without being destroyed or significantly repaired.

L19 ANSWER 34 OF 43 BIOSIS COPYRIGHT 2004 BIOLOGICAL ABSTRACTS INC. on STN
AN 1980:62471 BIOSIS
DN PREV198018062471; BR18:62471
TI ENHANCED **REACTIVATION** OF **UV IRRADIATED HERPES SIMPLEX** VIRUS INDUCED BY HUMAN CYTOMEGALOVIRUS.
AU NISHIYAMA Y [Reprint author]; RAPP F
CS PA STATE UNIV, COLL MED, HERSHEY, PA, USA
SO Abstracts of the Annual Meeting of the American Society for Microbiology, (1980) Vol. 80, pp. ABSTRACT 954.
Meeting Info.: 80TH ANNUAL MEETING, MIAMI BEACH, FLA., USA, MAY 11-16, 1980. ABSTR ANNU MEET AM SOC MICROBIOL.
CODEN: ASMACK. ISSN: 0094-8519.
DT Conference; (Meeting)
FS BR
LA ENGLISH

L19 ANSWER 42 OF 43 EMBASE COPYRIGHT 2004 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
AN 80084131 EMBASE
DN 1980084131
TI Enhanced survival of ultraviolet-**irradiated herpes** simplex virus in human cytomegalovirus-infected cells.
AU Nishiyama Y.; Rapp F.
CS Dept. Microbiol., Pennsylvania State Univ. Coll. Med., Hershey, Pa. 17033, United States
SO Virology, (1980) 100/1 (189-193).
CODEN: VIRLAX
CY United States
DT Journal
FS 047 Virology
LA English
AB Infection of primary **rabbit** kidney cells and Vero cells with human cytomegalovirus (HCMV) enhanced survival of **uv-irradiated herpes simplex** virus. In primary **rabbit** kidney (PRK) cells, maximal enhancement was observed when cells were preinfected with HCMV at multiplicities of 1 to 10 PFU/cell and when HCMV-infected cells were used to assay for **uv-irradiated HSV** within 2 days postinfection. HCMV-enhanced **reactivation** was sensitive to **uv-irradiation**, although virus **reactivation** was much more resistant to **irradiation** than infectivity. Furthermore, the enhancement process was sensitive to caffeine and pretreatment of cells with cycloheximide augmented the enhancement induced by HCMV infection.

L19 ANSWER 43 OF 43 LIFESCI COPYRIGHT 2004 CSA on STN
AN 88:58834 LIFESCI
TI Cutaneous **herpes** simplex virus lesions induced by ultraviolet **radiation**. A review of **model** systems and prophylactic therapy with oral acyclovir.
WELLCOME INTERNATIONAL ANTIVIRAL SYMPOSIUM PROCEEDINGS.
AU Spruance, S.L.; Lietman, P.S. [editor]; Fiddian, P. [editor]; Chapman, S.K. [editor]
CS Div. Infect. Dis., Dep. Med., Univ. Utah Sch. Med., Salt Lake City, UT 84132, USA
SO AM. J. MED., (1988) pp. 43-45.
Meeting Info.: Wellcome International Antiviral Symposium. Monte Carlo (Monaco). 2-4 Dec 1987.
DT Book

TC Conference
FS V
LA English
SL English
AB **Animal** and human **models** of ultraviolet **radiation**-induced **herpes simplex** virus disease provide opportunities to study the mechanism of virus latency and **reactivation**. These **models** can also be used to study the efficacy of antiviral agents. Prophylactic oral acyclovir altered the development of ultraviolet **radiation**-induced **herpes** labialis under both natural and experimental conditions.